

EXHIBIT C



PAUL J. MICHAELS, M.D.

**BOARD CERTIFIED IN ANATOMIC AND
CLINICAL PATHOLOGY, AND
CYTOPATHOLOGY**



Expert Report of Paul J. Michaels, M.D.
(Re: Sandra Childress)

BACKGROUND:

I am certified by the American Board of Pathology in Anatomic Pathology, Clinical Pathology, and Cytopathology. I attended and received my medical degree from the University of California, Los Angeles School of Medicine. I completed a residency in anatomic and clinical pathology at Massachusetts General Hospital, an affiliate of the Harvard School of Medicine, where I was a Clinical Fellow in Pathology. Following my residency, I completed a year of subspecialization in Cytopathology, also at Massachusetts General Hospital.

I am a pathologist affiliated with Clinical Pathology Associates in Austin, Texas. I have staff privileges at University Medical Center at Brackenridge, Seton Medical Center Austin, St. David's Medical Center, Seton Northwest Hospital, Seton Southwest Hospital, Seton Highland Lakes Hospital, Dell Children's Medical Center of Central Texas, Arise Austin Medical Center, Westlake Medical Center, Central Texas Medical Center (San Marcos, TX), and Resolute Health Hospital (New Braunfels, TX). Presently, I am the Laboratory Director for two separate Stat clinical laboratories in the Austin area, both affiliated with Clinical Pathology Laboratory/Sonic Healthcare USA, the third largest pathology company in the United States. During my career, I have had a strong subspecialty focus in breast and gynecology pathology, as well as cytopathology. I regularly attend and participate in tumor multidisciplinary conferences. In addition, I was a contracted speaker with Genomic Health, Inc., specializing in the *Oncotype DX* Breast Cancer Assay. I was also an invited speaker at the annual National Interdisciplinary Breast Center Conference for the National Consortium of Breast Centers, both in 2012 and 2013, lecturing on various topics in the field of breast cancer. My current curriculum vitae is attached to this report.

I have been asked to review medical records and evaluate slides prepared from the excised vaginal mesh pathology specimen from Sandra Childress. I also have reviewed other materials, including internal Ethicon documents, scientific literature, deposition testimony, and other materials in arriving at my findings and opinions in this case, a list of which is attached to my report. All of my opinions stated below are held to a reasonable degree of medical and scientific certainty and I reserve the right to modify or change my opinions based on further documents or information that may be provided to me in the future.

SUMMARY OF OPINIONS:

1. Polypropylene surgical mesh, including the Ethicon TVT device, elicits a chronic foreign body inflammatory reaction in tissue;
2. Certain design features of polypropylene surgical mesh, including the Ethicon TVT device, lead to scar bridging between polypropylene fibers and scar plating with encapsulation;
3. During repair of the tissue damaged by placement of the mesh device, the wound site contracts and shrinks the implant area;
4. Bridging fibrosis, scar plating/encapsulation, and shrinkage lead to a hardened, rigid device that is damaging to the surrounding vaginal mucosa;
5. The foreign body inflammatory reaction and resultant scarring and mesh contraction can lead to mesh-related complications like nerve entrapment (pain), erosion and extrusion, sexual pain, and urinary/bowel dysfunction.
6. Histopathological analysis of the pathological specimen of Ms. Childress' explanted TVT mesh demonstrated a prominent inflammatory and fibrosing reaction secondary to the mesh, including scar plate formation, bridging fibrosis, acute and chronic inflammation, erosion into the overlying urothelium, involvement of adjacent bladder muscular stroma, polypropylene bark degradation, and granuloma formation.
7. Ms. Childress' reported symptoms of right lower quadrant pain, recurrent stress urinary incontinence, and recurrent urinary tract infections were the direct result of these pathological features noted in my observations above.

COMMENT:

Not long after the commencement of transvaginal mesh (TVM) repair for POP and SUI, many complications were reported as being directly related to sequela from the host response of the implanted synthetic graft. The most common complications included mesh erosion and extrusion of the mesh through the vagina, pain, bleeding, secondary infection, dyspareunia, urinary problems, partner pain, and even organ fistula formation. Many of these complications required repeat surgical intervention. The pathologic response to the synthetic grafts used in surgery depends in large part on the physical and structural properties of the prosthesis. This host response varies based on mesh absorbability, pore size (size between filaments), and overall weight/density.

While absorbable material initially elicits a chronic foreign body inflammatory reaction, following complete absorption and subsequent fibroblast proliferation, the material is replaced by collagen-rich connective tissue, devoid of most acute or chronic inflammatory elements with obvious resolution of any foreign body reaction (Klinge 1999; Klinge 2001). In contrast, non-absorbable prosthetic material such as polypropylene is typically characterized by a persistent inflammatory response with ongoing foreign body-type giant cells (tissue macrophages), chronic inflammatory cells, and neovascularization. A study of modified mesh material used experimentally in a surgical setting that contained a mixture of non-absorbable polypropylene and absorbable polyglactin showed that reducing the amount of polypropylene (non-absorbable) to less than 30%, though still providing the necessary mechanical stability, significantly reduced the

degree of inflammation and subsequent fibrosis, leading to increased mesh flexibility (Klinge 1998). In a separate experimental study in dogs, also by Klinge and colleagues (1998), a multifilament combination of nonabsorbable polypropylene and absorbable polyglactin histologically showed considerably less inflammation and stromal fibrosis, compared to monofilament polypropylene grafts.

Studies over many years have uniformly supported the finding that larger mesh pore sizes have better incorporation into the surrounding native tissues (Greca 2001; Klinge 2002; Weyhe 2006). Whereas smaller pore sizes significantly impair vessel and adipose tissue penetration secondary to prominent fibrosis between adjacent mesh filaments (“bridging fibrosis”) (Chvapil 1969; Klinge 1999; Klinge 2002; Klosterhalfen 2005; Cobb 2006), larger-pore mesh material allows for infiltration by vascularized connective fibroadipose tissue (Taylor 1972; Cobb 2015) both structurally allowing for reduced fibrosis with subsequent retention of flexibility (Orenstein 2012; Lake 2015) and decreasing the ability for infection by bacteria introduced into the surgical site at the time of implantation (Merrit 1979). Additionally, the foreign body-type giant cell response and prominent fibrosis invariably encasing small pore meshes often forms a capsule surrounding the whole mesh (“scar plate”), resulting in the mesh becoming stiff, contracted, and nonflexible (Klosterhalfen 2005). Therefore, mesh contraction is defined by the reduction in surface area of the original implanted graft due to a retraction of the fibrotic scar tissue around the mesh.

Meshes with larger pore size generally have less material per square unit of measurement and are therefore of a lower weight, whereas those with smaller pore sizes are heavier. Analogous to the data showing a favorable host response with large pore size mesh, studies have concluded that light weight synthetic mesh shows better tissue integration with less inflammation and scar fibrosis (Klinge 1999; Klinge 2002; Klosterhalfen 2005), while the extent of stiffness increased directly in relationship to mesh weight (Cobb 2006).

In addition to the above described histological tissue responses and morphological modifications to implanted synthetic graft material, the clinical symptom of pain has become a significant postoperative complication in surgical cases using polypropylene mesh. The etiology of postsurgical pain can obviously be multifactorial, and several authors have evaluated the clinical consequence of pain in the context of patients with mesh grafts. In some instances, the marked mesh contraction secondary to bridging fibrosis and scar plate formation leads to erosion through the vaginal wall with a resulting acute inflammatory response, triggering regional pain. Similarly movement and shrinkage/kinking of the mesh may lead to migration towards nearby structures, ultimately causing fistulas, organ dysfunction, or even perforation. In contrast, more subtle histological features have also been shown to correlate with an increased sensation of pain. A study by Bendavid et al (2015) showed that mesh explants in patients complaining of pain contained a higher nerve density compared to tissue examined from patients who simply experienced a hernia recurrence. In this study, many of the nerves showed distortion and entrapment by the mesh material and fibrosis, while occasional areas resulted in the microscopic appearance of a marked neural proliferation, termed a “traumatic neuroma.” Therefore, in patients experiencing chronic pain and/or dyspareunia, studies showing significant resolution of

symptoms following removal of the mesh (Firoozi 2012; Crosby 2014; Danford 2015), support the idea that nerve proliferation and entrapment by fibrosis/scarring between the mesh filaments as a likely etiology.

With respect to the histologic features that accompany these functional properties, an Ethicon scientist, Dr. Joerg Holste (2005), noted that such meshes lead to excessive scar plate formation, while others, including Dr. Klinge and colleagues, found that the degree and quality of the fibrosis was directly related to the amount of the inflammatory reaction and associated foreign body reaction at the interface between the mesh and the patient's tissue. These cellular responses result in subsequent restriction of the graft, leading to significant complications of chronic pain. In addition, it is clear from Ethicon's internal documents that its polypropylene mesh products are associated with considerable mesh contraction resulting from the fibrous stromal reaction in their surgical meshes containing polypropylene (ETH.MESH.01774758). When mesh contracts or shrinks, it can cause the patient to experience complications, including scarring and chronic pain. According to Ethicon's Medical Affairs Director, Piet Hinoul, who testified in 2013 in *Gross et al vs Gynecare et al*, complications associated with its Prolift device include histological findings of a significantly scarred vagina with life-long risk of erosion, mesh contraction resulting in severe, chronic pain, and the presence of a severe, chronic inflammatory reaction to the mesh material in some patients resulting in the formation of a scar plate and/or bridging fibrosis. According to Ethicon's internal documents there is "significant evidence that the complications associated with synthetic meshes can cause significant morbidity including infection, erosion, exposure, and pain."

In a 2007 presentation prepared by Ethicon's Research and Development (R&D) department, it was concluded that for an ideal vaginal mesh to not ultimately result in a negative sexual impact for the patient, the graft material would ideally be lightweight and with a large pore size (ETH.MESH.01218361-01218367). With respect to the use of this product in the vaginal floor, Ethicon's internal documents demonstrate that "the vaginal tissue to be augmented is often structurally compromised, atrophic, and devascularized. Such poor tissue quality increased the risk of poor tissue incorporation into the mesh potentially resulting in suboptimal healing and mesh exposure or erosion into an adjacent viscous." This was further verified in 2008 by Dr. Klosterhalfen, the Head of the Duren Institute who was hired as an outside pathology consultant for Ethicon, who summarized his microscopic findings in these cases by noting that the "foreign body tissue reaction followed by secondary fibrosis seems to play a special role in pelvic floor repair" (ETH.MESH.00006636). He actually had informed Ethicon two years prior (2006) that, also based on his studies, the foreign body reaction to these meshes can occur for up to 20 years (ETH.MESH.00870466). Additionally in 2008, he went on to state that this inflammatory reaction is important "because soft tissue coverage is thin in pelvic floor repair" and "fibrosis and folding in this area induce mesh erosions and ulcerations." In a following report delivered the next year, Dr. Klosterhalfen reported, following his histological evaluation of an additional 172 prolapse mesh specimens, that "fibrosis inevitably leads to mechanical irritation, particularly when wrinkling occurs, and should be seen as the basic cause of mesh-induced erosion and ulceration," leading to a setting in which "infection is commonly observed following erosion in the vaginal mucosa" (ETH.MESH.02157879-02157880). Ethicon's documents demonstrate that

over the course of Dr. Klosterhalfen's interactions and meetings with Ethicon, he made numerous suggestions aimed at improving the biocompatible nature of mesh implants, including with regards to the choice and weight of the material used, the pore size, and the mechanical characteristics of the mesh products.

Finally, there are numerous publications and internal Ethicon documents that have demonstrated that polypropylene, including Ethicon's Prolene used to manufacture its SUI and POP mesh devices, undergoes *in vivo* degradation over time (Liebert 1976; Jongebloed 1986; Mary 1998; Costello 2007; Clave 2010; Wood 2013; ETH.MESH.15955438; ETH.MESH.1595540; ETH.MESH.15955463; ETH.MESH.13334286). After implantation of polypropylene, the inflammatory response to the foreign body causes an oxidative burst of free radicals and peroxides leading to embrittlement, crack formation, and loss of mechanical properties (Mary 1998). It has also been found that cholesterol and esterified fatty acids can diffuse into the amorphous zones of polypropylene and impact its physical and mechanical properties, causing damage to the surface (Clave 2010). In many cases, within the cracked and degraded surface layer, blue synthetic granules consistent with a blue pigment that Ethicon adds to the polypropylene resin during the manufacturing process to color some of the mesh fibers blue to aid in visibility can be seen. This finding rules out the possibility that the cracked surface layer is biological, a conclusion which was reached by Ethicon's own scientists in 1984 who used polarization light microscopy (ETH.MESH.15955462). Surface degradation of the polypropylene, including Ethicon's Prolene-based mesh devices, causes the device to become brittle and crack. This phenomenon increases the inflammatory and foreign body reaction and is a contributing cause of the complications experienced by patients (Mary 1998; Clave 2010).

SANDRA CHILDRESS:

Sandra Childress was born on 2/1/1961. She had 2 pregnancies, resulting in 2 deliveries. She reportedly admits to occasional cigarette use, though no substantial alcohol intake. Her past medical history is significant for bilateral ovarian cysts requiring multiple surgeries in her twenties. In April of 1991 she presented to her primary care provider with symptoms of stress urinary incontinence with evidence of both a Grade 2 cystocele and Grade 2 uterine prolapse. On 6/5/1991, she underwent a total abdominal hysterectomy with bladder suspension. Pathology from this surgery was predominantly unremarkable, except for the presence of a uterine leiomyoma (fibroid). In December of that same year, she again presented with symptoms of urinary incontinence. A few months later, on 2/12/1992, she was seen by Dr. Jim Hays with complaints of abdominal pain and stress urinary incontinence that had dramatically increased in severity. On examination, she was noted to have a Grade 2 cystocele with Valsalva (Grade 1 at rest).

In June of 2006, Ms. Childress again presented to Dr. Hays with worsening urinary leakage with walking and movement with an urge component. On examination during this visit, she had a Grade 2 cystourethrocele. Later that month, on 6/30/2006, Dr. Hays performed a sling procedure with TVT placement, done through an abdominal route, at Fort Sanders Regional Medical Center.

Although she initially seemed to do well, she again presented to Dr. Hays on 7/30/2007 with right-sided pelvic pain for 3-4 months and associated incomplete bladder emptying and urinary leakage. An abdominal/pelvic CT scan performed on 8/2/2007 showed a thickened right urinary bladder wall. Follow-up cystoscopic evaluation on 8/14/2007 showed exposed mesh from the 7 to 11 o'clock region of the bladder mucosal surface. Following this procedure, Dr. Edward Tieng at TUA Urology Associates of Knoxville performed a repeat cystoscopy with excision of the mesh within the bladder wall. Approximately six weeks later, on 10/3/2007 at Fort Sanders Regional Medical Center, Drs. Tieng and Flickinger performed a pelvic exploration with sling procedure, cystotomy, and excision of TVT sling. Although Ms. Childress had a urinary tract infection in May of 2009 (Positive for *Escherichia coli*), she has done relatively well since without gynecologic or genitourinary complaints.

MICROSCOPIC FINDINGS:

FINAL DIAGNOSIS:

1). BLADDER MESH AND SOFT TISSUE, EXCISION:

Synthetic material (consistent with mesh) with associated perifilamentous (“bridging”) and encapsulating (“scar plate”) fibrosis, erosion through the genitourinary urothelium with involvement of the underlying bladder smooth muscle stroma, acute and chronic inflammation, polypropylene bark degradation, and a foreign body giant cell response.

As part of my review in this case, I received four microscopic slides of tissue specimens surgically removed from Ms. Childress on 10/3/2007. Two of these slides were stained with Hematoxylin and Eosin (H&E) and two were unstained. The histologic sections show fragments of fibromuscular tissue, some of which are surfaced by a layer of focally denuded and eroded urothelium. The urothelium has a stratified lining and shows reactive, papillary hyperplasia with an edematous lamina propria containing congested vessels with evidence of hemorrhage and a neutrophilic infiltration. Mesh filaments are present that involve the urothelial surface with evidence of erosion and extrusion, with surrounding reactive fibrosis, including both bridging fibrosis and dense, hyalinized fibrous tissue that encases the mesh structure (“scar plate” fibrosis). Also noted associated with the mesh filament spaces is a chronic inflammatory reaction containing numerous lymphocytes and plasma cells, including scattered foreign body multinucleated giant cells. The fibroinflammatory reaction surrounding the mesh involves the adjacent bladder smooth muscle bundles. On polarization light microscopy, residual polypropylene is noted, including areas of bark that show varying degrees of degradation with associated cracking.

Within the tissue I examined from Ms. Childress’ mesh explant, I identified many of the histological findings that have been described in association with transvaginal mesh grafts and that also correlate with her reported symptomatology.

Inflammation is a complex tissue reaction to injurious agents resulting in vascular responses, migration and activation of leukocytes, and, occasionally, systemic consequences. Inflammation is divided into acute and chronic patterns.

Acute inflammation has three major components including alteration in vascular caliber and blood flow, structural changes in the microvasculature that permit plasma proteins and leukocytes to leave the circulation, and emigration of leukocytes, mainly neutrophils, from the microcirculation to the focus of injury. Acute inflammatory reactions can be triggered by a variety of stimuli, including foreign bodies. Vasodilation is one of the principle manifestations of acute inflammation which results in increased blood flow that often manifests as an area of increased heat and redness. Clinically, these features not infrequently lead to a complaint of pain at the site of increased blood flow. Within Ms. Childress' mesh explant tissue, I identified several areas of vascular dilatation and congestion (Figure 1) that were present associated with the overall fibrotic tissue response. These areas of vascular congestion with associated tissue edema within the mesh pores are indicative of a localized and compartmentalized anatomic environment resulting from the compressed graft framework, potentially leading to inflow/outflow abnormalities within the local circulation (Bendavid 2015). These effects on the vascular supply to the region can lead to ischemic changes that result in clinical signs and symptoms that Ms. Childress experienced. A consequence of a more significant inflammatory response, in addition to vasodilation and congestion, is direct endothelial injury, resulting in endothelial cell necrosis and detachment. These findings are most often associated with extravascular hemorrhage into the surrounding stroma, as seen in Ms. Childress' mesh explant tissue (Figure 2).

Classically, the presence of neutrophils is the hallmark of acute inflammation within tissue. Once neutrophils have migrated from the vascular space into the tissue, they become activated leading to the production of arachidonic acid metabolites, degranulation and secretion of lysosomal enzymes, activation of the oxidative burst, and secretion of cytokines, all processes which contribute to regional pain at the site of inflammation/injury. Within Ms. Childress' mesh explanted tissue, foci of neutrophils were identified (Figure 3), again correlating with her clinical complaint of pain. It would be expected that the inherently destructive nature of the acute inflammatory response would need controls to minimize the extent of tissue damage. Generally, the degree of inflammation declines as the mediators of inflammation have short half-lives and are degraded after being released. However, in the case of a persistent stimulus, such as a non-absorbable mesh, in a compromised anatomic region, such as the pelvic floor, the ongoing contraction and migration of the foreign body can lead to continued tissue injury with associated clinical sequelae such as pain.

Chronic inflammation is considered to be an inflammatory reaction in which active inflammation, tissue destruction, and attempts at repair are occurring concurrently within a defined region. Like acute inflammation, there are numerous etiologies for chronic inflammation, again including prolonged exposure to potentially toxic endogenous or exogenous agents. An example of such an exogenous agent would be non-absorbable polypropylene mesh. In contrast to acute inflammation, which is manifested by various vascular changes and a predominantly neutrophilic infiltration, chronic inflammation is characterized by involvement by mononuclear

cells, mainly lymphocytes and macrophages, and connective tissue replacement of damaged tissue. The macrophage is typically the dominant cell noted in the context of a chronic inflammatory reaction. Macrophages, once activated, like neutrophils, secrete a wide variety of biologically active products. However, in the case of macrophages, this cascade of events leads to the recruitment of other inflammatory cells, namely lymphocytes, which can ultimately result in tissue fibrosis. The bidirectional interaction of lymphocytes and macrophages together is characteristic of chronic inflammatory responses, as macrophages display foreign antigens to T lymphocytes, which ultimately stimulates T-cell responses. The chronic inflammatory response is typically the dominant type of local immune reaction seen in foreign body reactions, such as to synthetic mesh. Examination of Ms. Childress' explanted mesh tissue clearly confirms evidence of this chronic inflammatory reaction, characterized by increased numbers of lymphocytes and macrophages within the tissue (Figures 4 and 5).

Granulomatous inflammation is a distinctive pattern of a chronic inflammatory reaction encountered in a limited number of immunologically mediated, infectious and non-infectious conditions. One type of granuloma, termed a foreign body granuloma, as the name implies develops when foreign material is introduced into the tissue and is large enough to preclude phagocytosis by a single macrophage. This reaction is often characterized by the presence of multinucleated giant cells, representative of fused epithelioid macrophages. Evidence of this foreign body granulomatous response is clearly observed histologically in the microscopic sections taken from Ms. Childress' explanted mesh (Figures 6 and 7).

The chronic inflammatory response is closely intertwined with the process of repair. During repair, the injured tissue is replaced through regeneration of native parenchymal cells, by filling of the defect with fibrous tissue (scarring) or, most commonly, by a combination of these two processes. Tissue repair by fibrosis, though an attempt at healing with subsequent strengthening and repair of the tissue, may be harmful depending on the degree of collagen deposition and anatomic location in which it occurs. In a broad sense, the term "fibrosis" applies to any abnormal deposition of connective tissue, though the degree of such deposition will determine the functional impairment, if any. When large defects are initially present, or in the case of some larger foreign bodies, a greater degree of granulation tissue is formed and subsequent wound contraction can occur. Fibrotic bridging is a histological phenomenon closely associated with the clinical consequence of mesh shrinkage. Fibrotic bridging refers to collagen deposition and inflammatory cell infiltration exceeding more than half of the pore size of the mesh (Klinge 2002). A significant degree of "bridging fibrosis" and "scar plate" formation was noted histologically in the microscopic sections from Ms. Childress' explanted specimen (Figures 8-10). These findings are consistent with mesh shrinkage whereby the tissue contracts during the healing process, pulling with it the synthetic mesh device which can damage nerves that come into contact with mesh fibers or become entrapped within the mesh and/or mesh scar complex leading to pain and other complications. Of note within this case, the fibroinflammatory response associated with Ms. Childress' mesh specimen involves the underlying smooth muscle bundles of the bladder region (Figures 11-13) and shows evidence of erosion through the urothelium of the genitourinary tract (Figures 14 and 15). Both of these histological findings correlate well with her report of genitourinary symptoms of recurrent stress incontinence and urinary tract infections.

Like Ethicon's scientists, using standard histopathology techniques, I found that the surface layer of the TVT device that was implanted in Ms. Childress for nearly 1 1/2 years degraded while implanted in her body. Using polarization light microscopy, I observed that the surface of the mesh fibers was cracked, extending to the central core of the mesh space (Figures 16 and 17). The brittleness of this degraded polypropylene implant can impact the biocompatibility of the device and result in patient complications like those experienced by Ms. Childress, including right lower quadrant pain, inflammation, scarring, and organ dysfunction (Mary 1998; Clave 2010).

FIGURES:

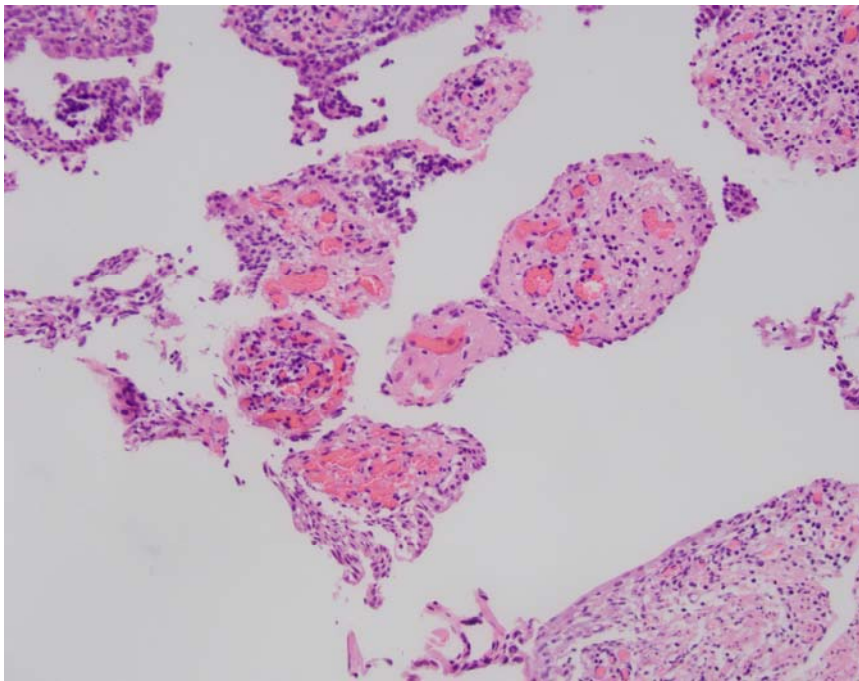


Figure 1: Eroded urothelium adjacent to extruded mesh material showing prominent stromal vascular congestion (200x magnification).

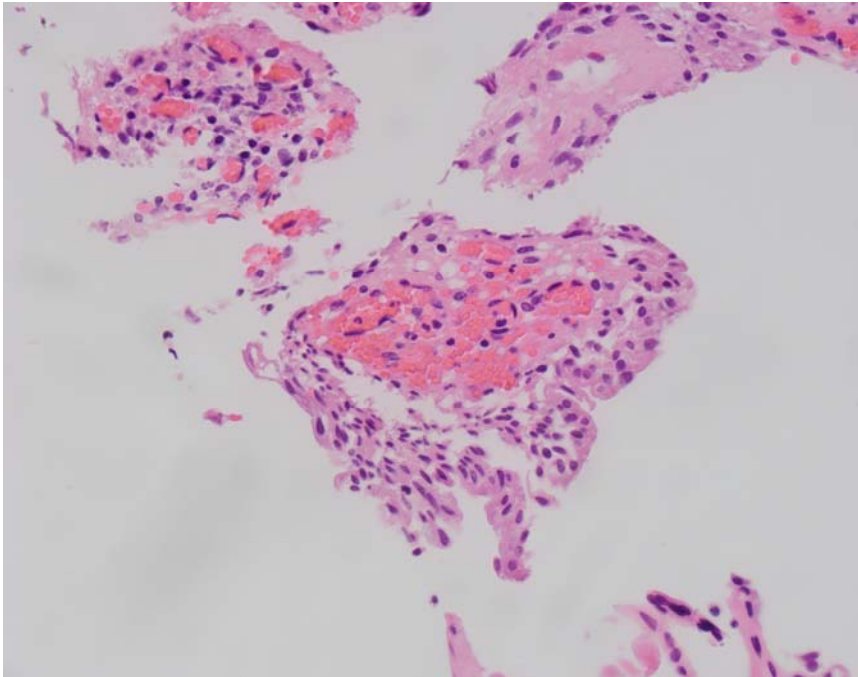


Figure 2: Areas of red blood cell extravasation/hemorrhage within the supporting stroma of the urethral tissue beneath the epithelium and nearby the extruded mesh (400x magnification).

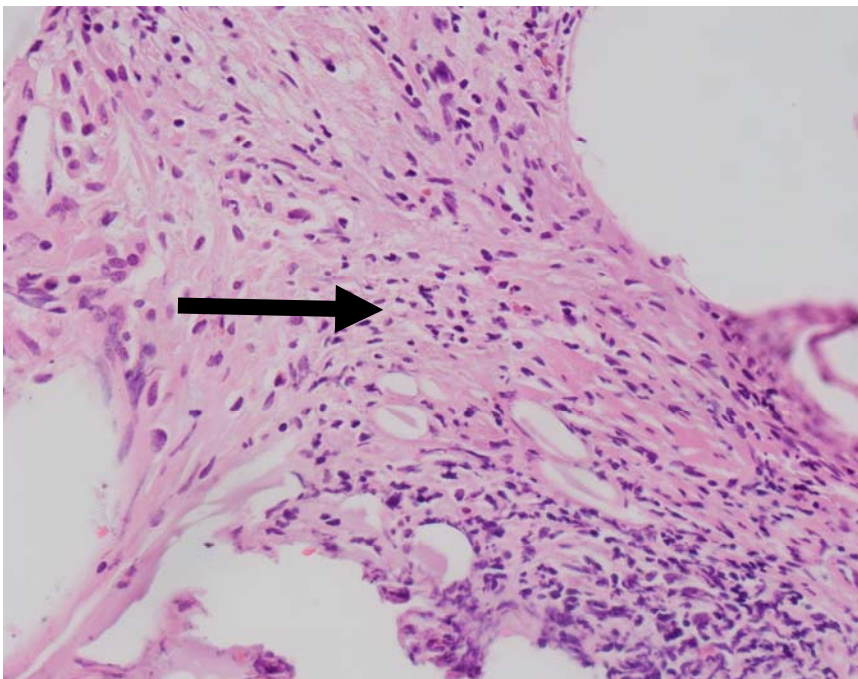


Figure 3: Neutrophil infiltration (arrow) associated with a background of chronic inflammation and fibrosis, adjacent to mesh filaments (400x magnification).

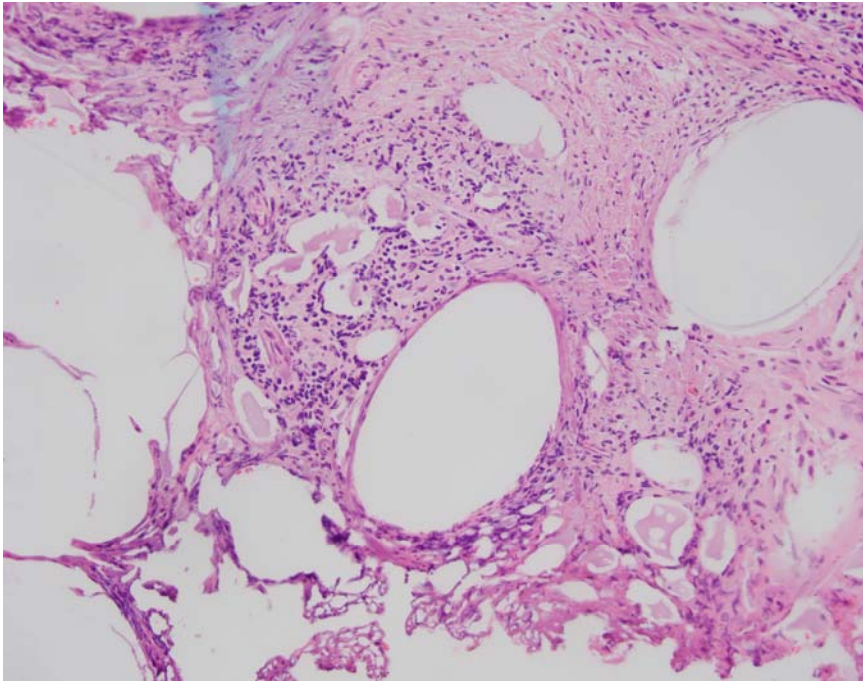


Figure 4: Mesh filaments surrounded by a chronic inflammatory response including numerous lymphocytes and macrophages (200x magnification).

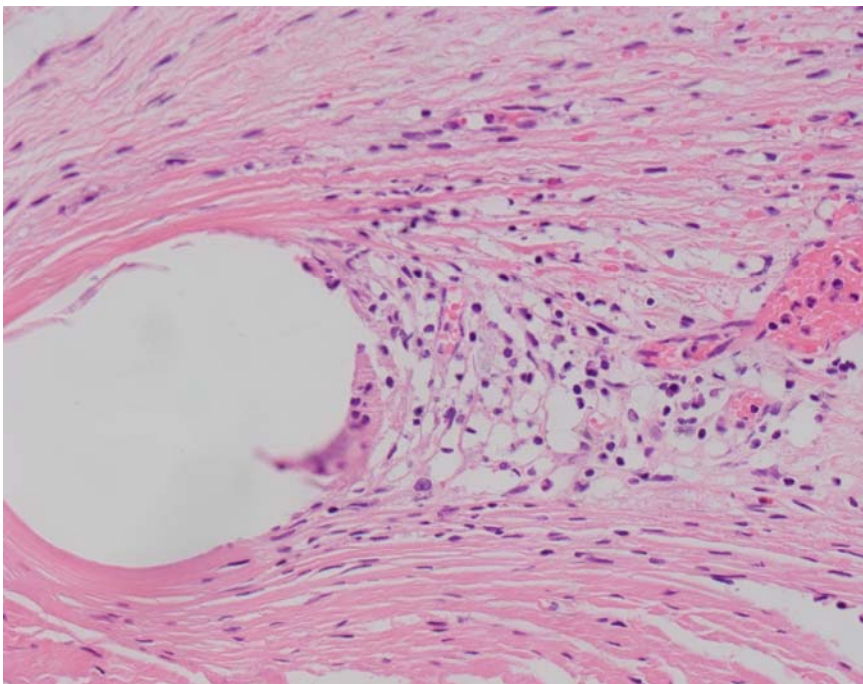


Figure 5: Lymphocytes and macrophages (chronic inflammation) present adjacent to a mesh filament and surrounded by fibrosis (400x magnification).

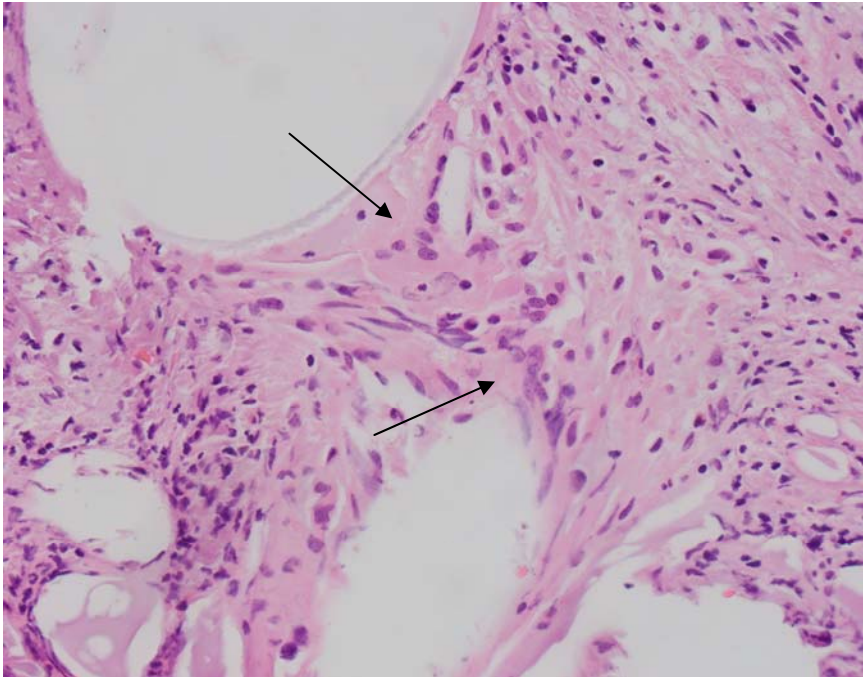


Figure 6: Multinucleated foreign body giant cells (arrows) surrounded mesh filaments and accompanied by a chronic inflammatory tissue reaction (400x magnification).

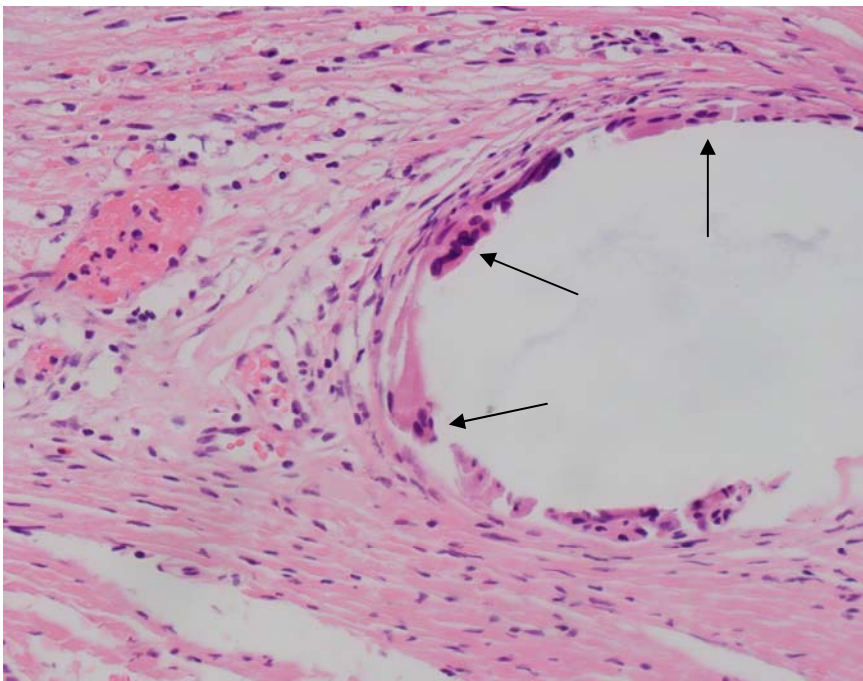


Figure 7: Multinucleated foreign body giant cells (arrows) surrounded mesh filaments and is accompanied by a chronic inflammatory tissue reaction (400x magnification).

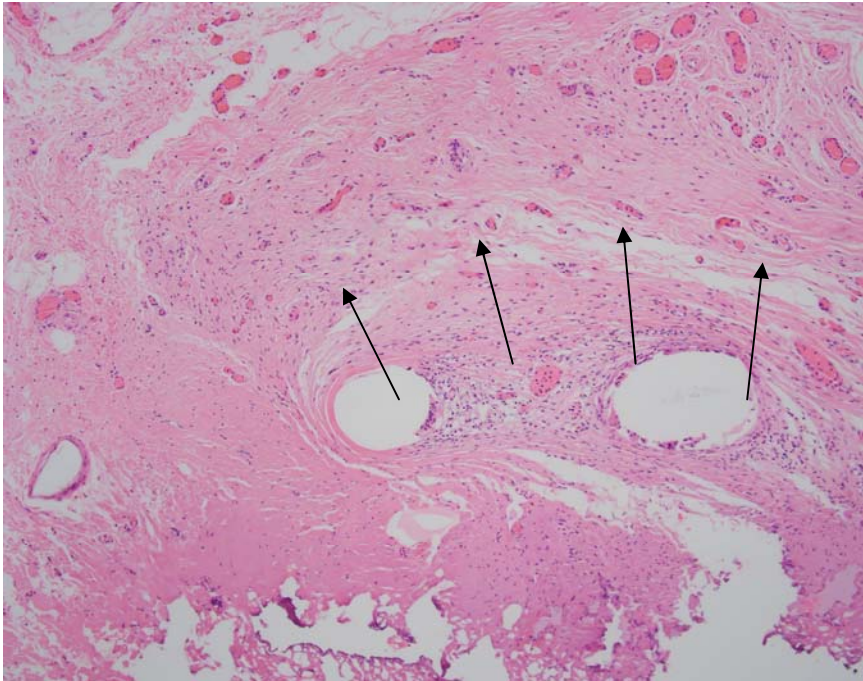


Figure 8: "Scar plate" (arrows) seen encircling/encapsulating mesh filaments (100x magnification).

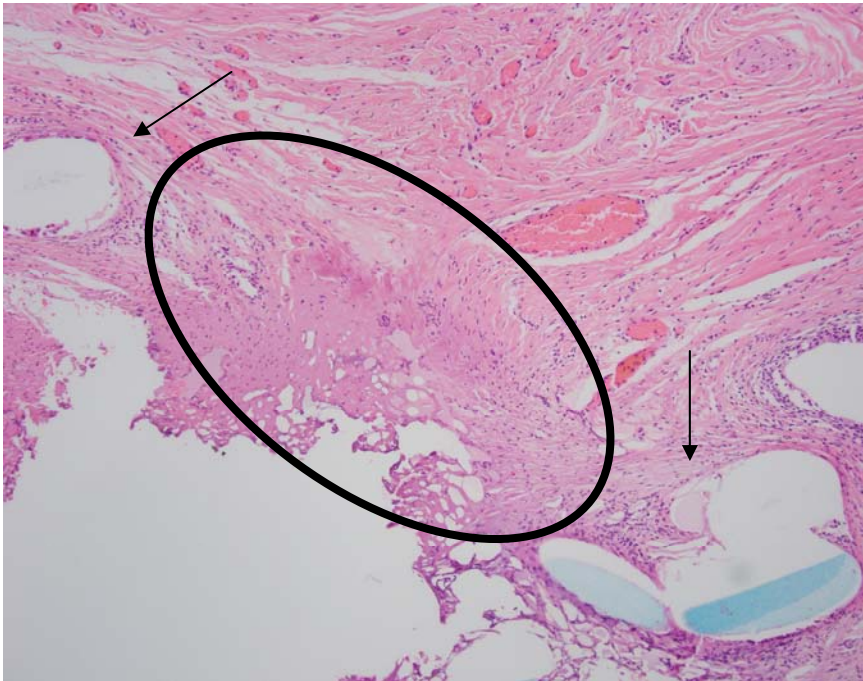


Figure 9: Area of bridging fibrosis (circle) separating mesh filaments (arrows) (100x magnification).

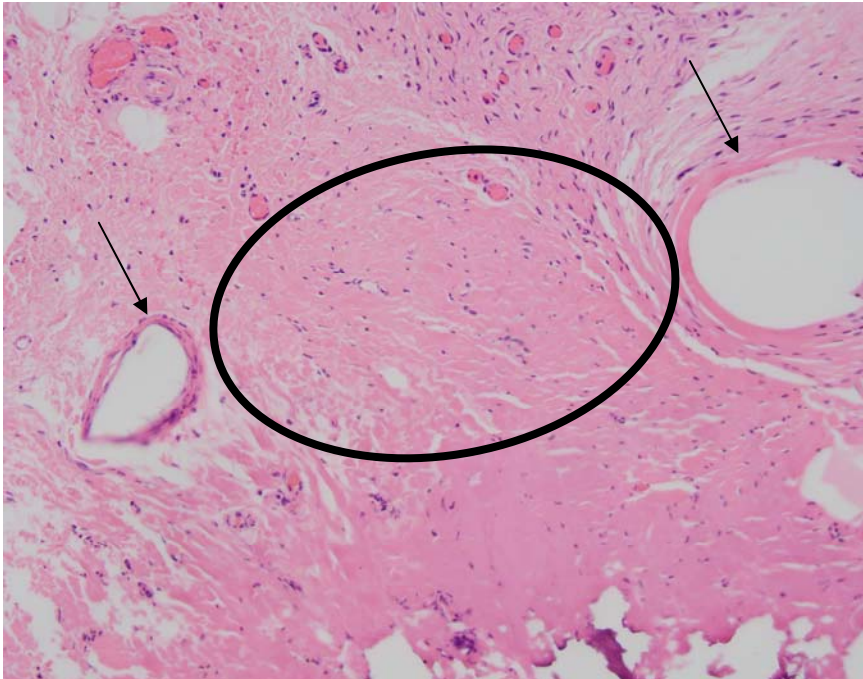


Figure 10: Area of bridging fibrosis (circle) separating mesh filaments (arrows) (200x magnification).

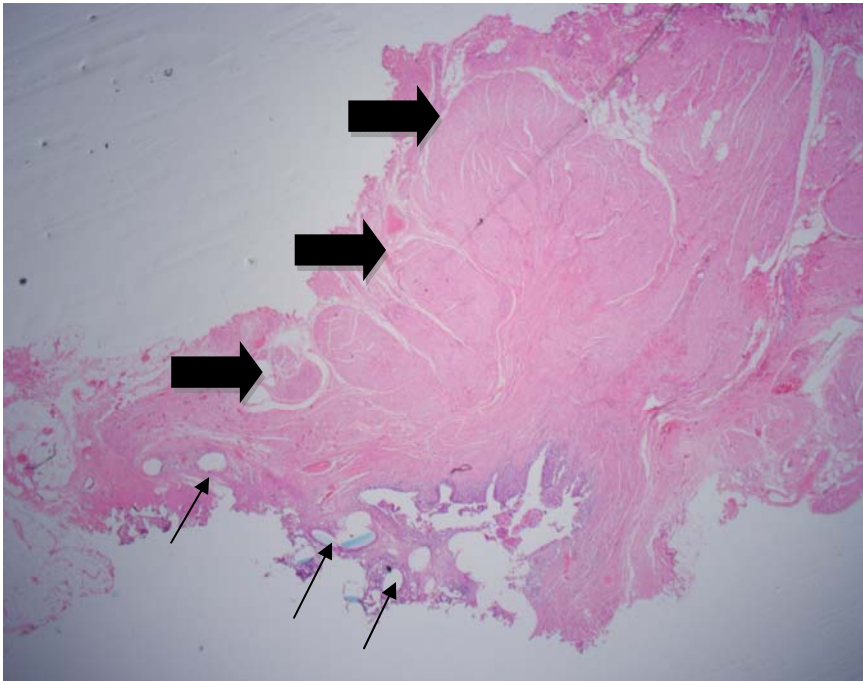


Figure 11: Low magnification appearance of Ms. Childress' specimen showing a fragment of mesh (thin arrows) with associated fibrosis involving the bladder smooth muscle bundles (block arrows) (20x magnification)

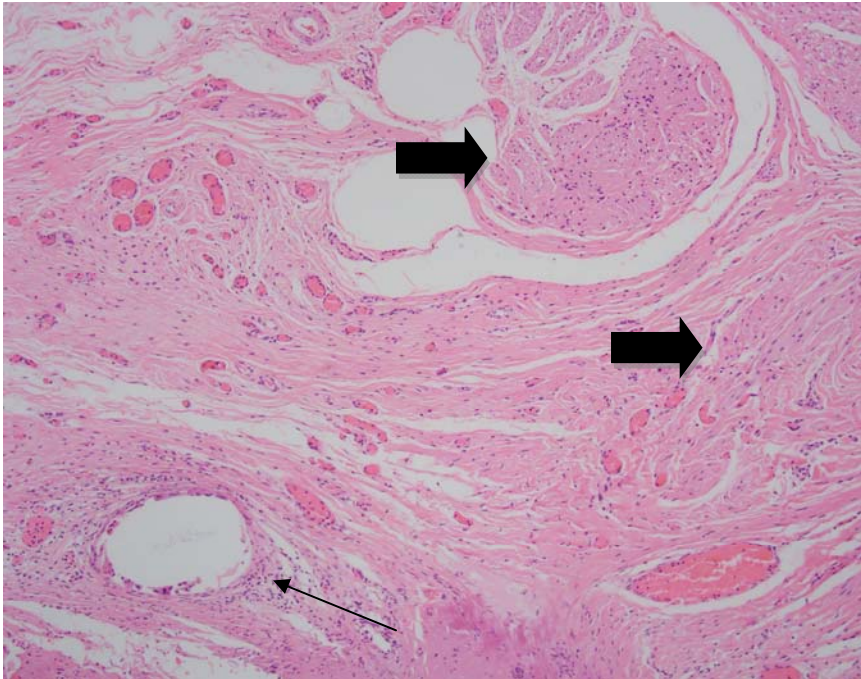


Figure 12: Mesh filament (thin arrow) with associated perifilamentous fibrosis and chronic inflammation, involving the bladder smooth muscle bundles (block arrows) (100x magnification).

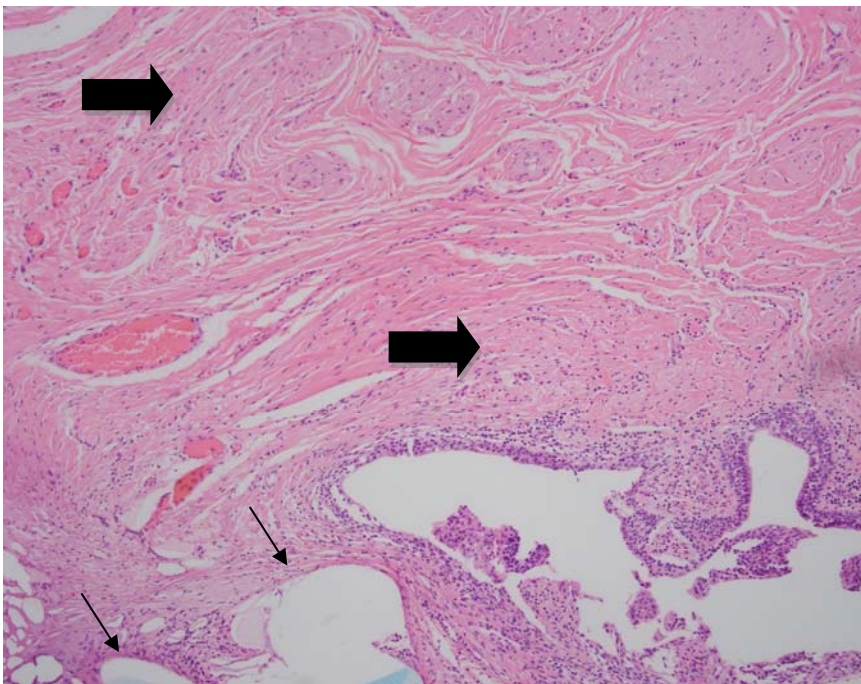


Figure 13: Mesh filaments (thin arrows) with associated perifilamentous fibrosis and chronic inflammation, involving the bladder smooth muscle bundles (block arrows) (100x magnification).

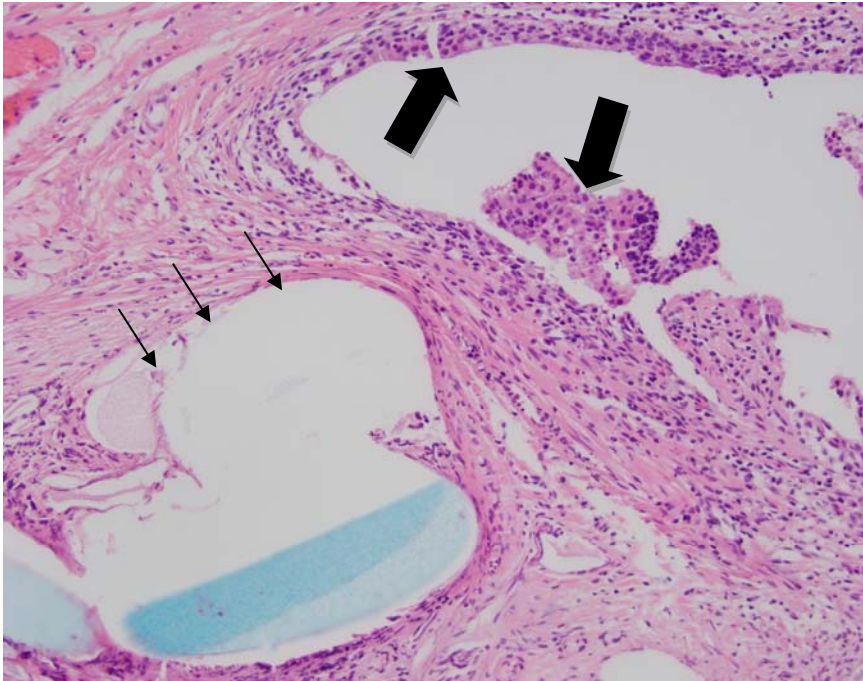


Figure 14: Mesh filaments (thin arrows) with associated perifilamentous fibrosis and chronic inflammation, eroding and extruding through the urothelial lining (block arrows) (200x magnification).

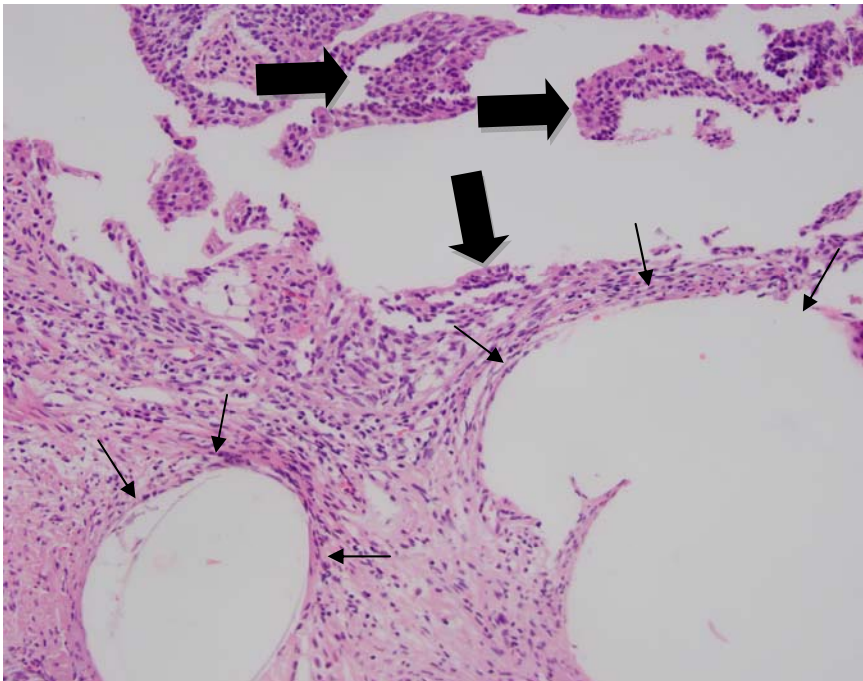


Figure 15: Mesh filaments (thin arrows) with associated perifilamentous fibrosis and chronic inflammation, eroding and extruding through the urothelial lining (block arrows) (200x magnification).

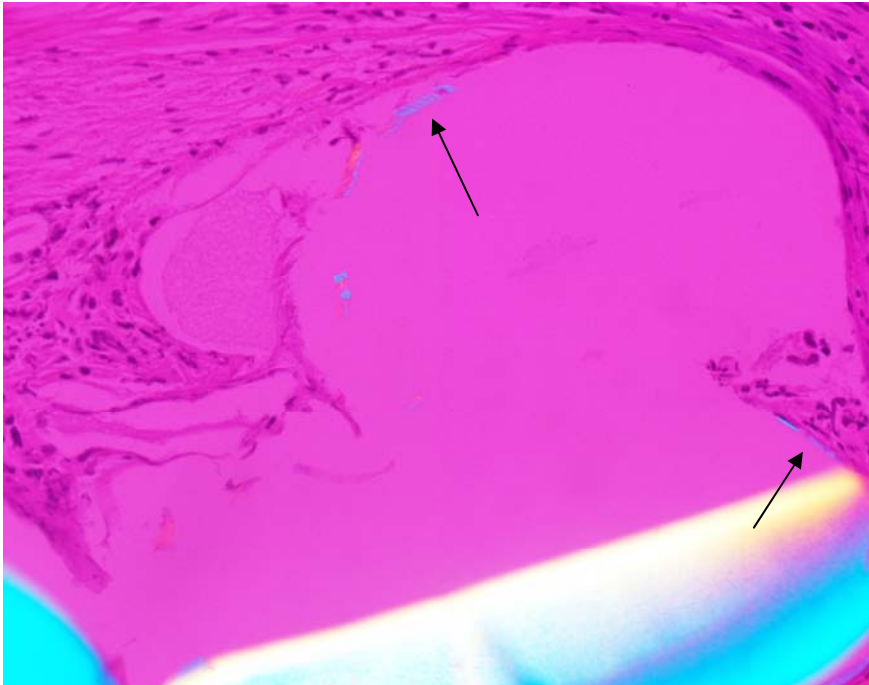


Figure 16: Polarization light microscopy showing residual and degraded polypropylene bark with numerous cracks (arrows) (400x magnification).

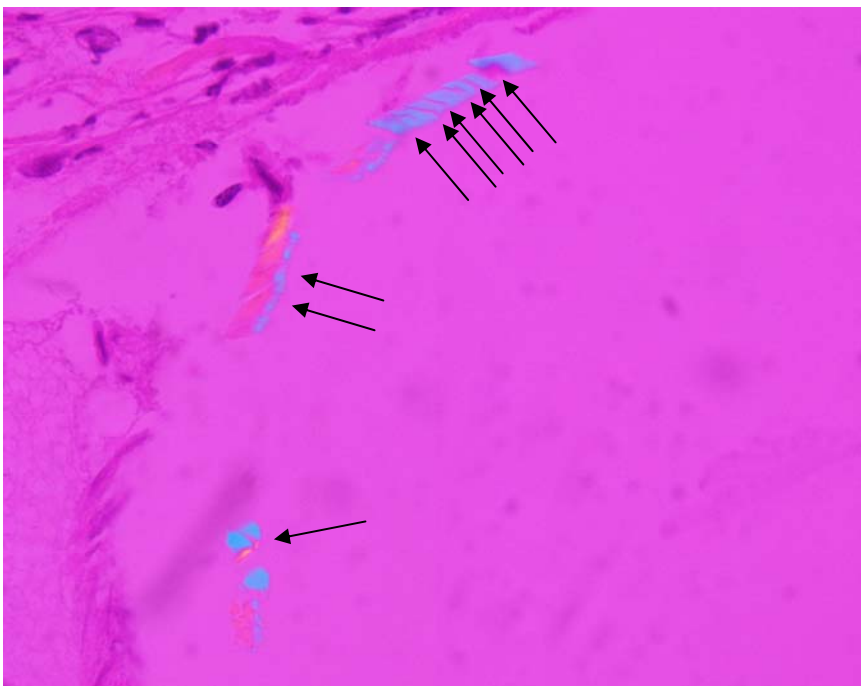


Figure 17: Polarization light microscopy under oil immersion showing residual and degraded polypropylene bark with numerous cracks (arrows) (1000x magnification).

SUMMARY:

In summary, based upon a detailed microscopic evaluation of Ms. Childress' explanted mesh specimen, it is my opinion to a reasonable degree of medical and scientific certainty that her reported symptoms of right lower quadrant pain, recurrent urinary tract infections, and stress urinary incontinence were the direct result of a prominent inflammatory and fibrosing reaction secondary to the synthetic mesh, including scar plate formation, bridging fibrosis, acute and chronic inflammation, extrusion and erosion into the overlying urothelium, involvement of adjacent bladder muscular stroma, polypropylene bark degradation, and granuloma formation.

All of the opinions I have expressed in this report are based on a combination of my personal experience as a diagnostic anatomic pathologist, my frequent interactions with clinical colleagues in the day to day management of patients, past and ongoing extensive review of pertinent literature in the field, various internal Ethicon documents, my review of the provided slides from Ms. Childress' excised mesh specimen, and the available medical records in this case. I express these opinions with a reasonable degree of medical certainty.

5/4/2016

DATE



Paul J. Michaels, M.D

EXHIBIT A

PAUL J. MICHAELS, M.D.

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PROFESSIONAL EXPERIENCE

February 2013 – Present

Pathologist at Clinical Pathology Associates

Austin, TX

- Member of ~40 pathologist group that covers several major hospitals in the Central Texas region (including the Austin area and surrounding communities, San Marcos, and San Antonio)
- Anatomic pathology responsibilities include frozen sections, sign-out of complex surgical specimens, review of numerous outpatient biopsies, GYN and NON-GYN cytology, presentation at tumor board conferences, and performing hospital autopsies
- Clinical pathology responsibilities include peripheral blood smear and body fluid review, crystal identification, assessment of blood transfusion reactions and RBC antibody serology work-ups, TEG analysis, and evaluation of protein electrophoresis panels
- Director of the Fine Needle Aspiration Clinic at University Medical Center, Brackenridge (February 2013 – Present)
- Medical Director of North Austin CPL Stat Laboratory (April 2013 – Present)
- Medical Director of South Austin CPL Stat Laboratory (January 2014 – Present)
- College of American Pathologists (CAP) Inspections:
 - Team Leader (June 2014)

July 2006 – January 2013

Pathologist at Laboratory Medicine Consultants/Aurora Diagnostics

Las Vegas, NV

- Member of a ~18 pathologist group that covered several hospitals in the southern Nevada and northwestern Arizona region
- Anatomic pathology responsibilities include frozen sections, sign-out of complex surgical specimens, review of numerous outpatient biopsies, GYN and NON-GYN cytology, coverage of outpatient FNA clinic, presentation at numerous tumor board conferences, and performing hospital autopsies
- Clinical pathology responsibilities include peripheral blood smear and body fluid review, crystal identification, assessment of blood transfusion reactions, protein electrophoresis and immunofixation analysis, and interpretation of various chemistry, lipid, coagulation, and serologic laboratory test panels
- Medical Director of MountainView Hospital Laboratory (January 2010 – January 2013):
 - Oversight of daily laboratory operations (~270 bed hospital)
 - Annual review of all laboratory policies
 - Member of the hospital Medical Executive Committee and Quality Council
- Laboratory Director of various ambulatory surgery centers, including:
 - Durango Surgery Center (February 2009 – January 2013)
 - Tenaya Surgery Center (January 2009 – January 2013)
 - Stonecreek Surgery Center (June 2008 – January 2013)
 - Las Vegas Regional Surgery Center (June 2007 – December 2007)
- Cytopathology Laboratory Director (August 2007 – January 2010)
 - Provided daily oversight of our centralized cytology laboratory for over 1,000 inpatient beds and numerous outpatient clinics in the surrounding community
 - Reviewed quarterly QA statistics, including NON-GYN/Surgical specimen correlation cases

- Cancer Conference Coordinator (January 2007 – December 2009) and Cancer Program Activity Coordinator (May 2007 – December 2009)
 - Reviewed all monthly cancer cases to assure compliance with Commission on Cancer standards
 - Provided oversight for all hospital Tumor Board conferences, including organization of the annual cancer conference assignments for all pathologists in the group
 - Attended bimonthly Sunrise Hospital Cancer Committee meetings
- College of American Pathologists (CAP) Inspections:
 - Team Leader (September 2009, August 2010)
 - Team Member (March 2007, September 2010)
- In 2012, was voted a “Top Doctor” in Pathology in Las Vegas, NV by Consumers’ Checkbook of Washington, D.C., published in *Vegas Seven* magazine (2/23/2012).
 - Received the most mentions of any pathologist in the city

October 2004 – June 2006

- Locum Tenens Pathologist at North Shore Pathologists Salem, MA
- Independently performed autopsies that occurred during the weekends and holidays
 - Prepared and signed-out (cosigned) the autopsy reports with an attending/supervising pathologist

EDUCATION/CLINICAL TRAINING

July 2005 – June 2006

Cytopathology Fellowship, Massachusetts General Hospital/Harvard Medical School

June 2001 – June 2005

- Anatomic/Clinical Pathology Residency, Massachusetts General Hospital/Harvard Medical School
- Chief Resident, Anatomic Pathology (June 2004 – November 2004)
 - Resident Representative for Mentoring, American Society of Cytopathology, Ethics and Conduct Committee (June 2004 – June 2006)

August 1996 – June 2001

- Doctorate of Medicine, University of California, Los Angeles
- Post Sophomore Fellowship in Anatomic/Clinical Pathology, Combined UCLA/Cedars Sinai Program (June 1998 – June 1999)
 - Alpha Omega Alpha Honor Society (Elected 2001)

September 1992 – September 1995

- Bachelor of Science, University of California, Irvine
- Major in Biological Sciences with a Minor in Microbiology, *Cum Laude*
 - Phi Beta Kappa Honor Society (Elected 1995)
 - UC Regents Scholar (1992 – 1995)

ACADEMIC APPOINTMENTS

June 2009 – June 2013

Adjunct Associate Professor of Pathology, Touro University of Nevada, College of Osteopathic Medicine

June 2001 – June 2006

Clinical Fellow in Pathology, Massachusetts General Hospital/Harvard Medical School

MEDICAL LICENSURE AND CERTIFICATION

October 2006 – Present

American Board of Pathology, Cytopathology (Time Limited, Recertified - March, 2015)

August 2005 – Present

American Board of Pathology, Anatomic and Clinical Pathology (Time Unlimited)

December 2012 – Present

Texas Medical Board (License # P5108)

December 2012 – Present

California Medical Board (License # C55645)

August 2006 – Present

Utah Medical Board (License # 6228782-1205)

June 2006 – Present

Nevada Medical Board (License #11907)

June 2006 – Present

Arizona Medical Board (License #35644)

October 2004 – June 2007

Massachusetts Medical Board (License #223088)

PROFESSIONAL ORGANIZATIONS

Texas Medical Association (TMA)

Travis County Medical Society (TCMS)

Texas Society of Pathologists (TSP)

American Society of Cytopathology (ASC)

American Society of Clinical Pathology (ASCP)

College of American Pathologists (CAP)

United States and Canadian Academy of Pathology (USCAP)

ACADEMIC PUBLICATIONS

Pusztaszeri M, Wang H, Cibas ES, Powers CN, Bongiovanni M, Ali S, Khurana KK, **Michaels PJ**, and Faquin WC. Fine-needle Aspiration Biopsy of Secondary Neoplasms of the Thyroid Gland: a Multi-institutional Study of 62 Cases. *Cancer Cytopathol* 2015;123:19-29.

Lewis Jr. BA, Zebrowski B, Yumiaco NS, **Michaels P**, and Erling M. Case Report of Paratesticular Liposarcoma with Metachronous Large Renal Cell Carcinoma. *Curr Urol* 2010;4:162-163.

Pitman MB, **Michaels PJ**, Deshpande V, Brugge WR, and Bounds BC. Cytological and Cyst Fluid Analysis of Small (<3 cm) Branch Duct Intraductal Papillary Mucinous Neoplasms Adds Value to Patient Management Decisions. *Pancreatology* 2008;8:277-84.

Michaels PJ, Brachtel EF, Bounds BC, Brugge WR, and Pitman MB. Intraductal Papillary Mucinous Neoplasms (IPMN) of the Pancreas: Cytologic Analysis and Correlation with Histologic Grade. *Cancer* 2006;108:163-73.

Steele DJ and **Michaels PJ**. Case Records of the Massachusetts General Hospital. Weekly Clinicopathological Exercises. Case 40-2004- A 42-year-old Woman with Long-Standing Hematuria. *N Engl J Med* 2004;351:2851-9.

Michaels PJ, Espejo ML, Kobashigawa J, Alejos JC, Burch C, Takemoto S, Reed EF, and Fishbein MC. Humoral Rejection in Cardiac Transplantation: Risk Factors, Hemodynamic

Consequences and Relationship to Transplant Coronary Artery Disease. *J Heart Lung Transpl* 2003;1:58-69.

Michaels PJ, Fishbein MC, and Colvin RB. Humoral Rejection in Human Transplantation. *Springer Semin Immunopathol* 2003;25:119-140.

Marchevsky AM, Lau SK, Khanafshar I, Ockhart C, Phan A, **Michaels PJ**, and Fishbein MC. Internet Teleconferencing Method for Telepathology Consultations from Lung and Heart Transplant Patients. *Hum Pathol* 2002;33:410-4.

Michaels PJ, Kobashigawa J, Laks H, Azarbal A, Espejo ML, Chen L, and Fishbein MC. Differential Expression of RANTES Chemokine, TGF- β , and Leukocyte Phenotype in Acute Cellular Rejection and Quilty B Lesions. *J Heart Lung Transpl* 2000;20:407-16.

Michaels PJ, Kobashigawa J, Child JS, and Fishbein MC. Chronic Right Sided Myocarditis Mimicking Arrhythmogenic Right Ventricular Dysplasia. *Hum Pathol* 2000;31:618-21.

Marchevsky A, Lockhart C, Phan A, **Michaels PJ**, and Fishbein MC. Web-Based Teleconferencing Techniques as Inexpensive Tools for Transplant Patients. *Lab Invest* 2000;80:37A.

Michaels PJ and Mautz WJ. Effects of Inhaled Ozone and Formaldehyde on Tracheal Epithelial Secretion of Rats Exposed During Rest and Exercise. *Journal of Undergraduate Research in the Biological Sciences* 1995;25:779-90.

PRESENTATIONS

INVITED TALKS:

April 2015

“*The Surgical Pathology of Dysphonia*” for the Masters Program in Speech and Language Pathology at University of the Pacific, Stockton, California.

January 2014

“*Cytology From Sin City*” at University of Colorado, Denver, Department of Pathology and Laboratory Medicine. Invited Grand Rounds Speaker.

“*Cytology Jeopardy*” at University of Colorado, Denver, Department of Pathology and Laboratory Medicine. Invited Unknown Conference for Residents.

March 2013

“*Confounding Metastatic Breast Cancer Controversy*” presented at the 23rd Annual National Interdisciplinary Breast Center Conference for the National Consortium of Breast Centers. Planet Hollywood Resort & Casino. Las Vegas, Nevada.

March 2012

Panelist for “*Interesting Cases: What Would You Have Done?*” presented at the 22nd Annual National Interdisciplinary Breast Center Conference for the National Consortium of Breast Centers. Paris Las Vegas Hotel & Casino. Las Vegas, Nevada.

November 2010

“*Cytology From Sin City 2*” at Massachusetts General Hospital and Brigham and Women’s Hospital, Departments of Pathology, Harvard Medical School.

February 2009

“*Cytology From Sin City*” at Massachusetts General Hospital, Brigham and Women’s Hospital, and Beth Israel Deaconess, Departments of Pathology, Harvard Medical School.

December 2003

"Thin Basement Membrane Nephropathy and Alport Syndrome" at Massachusetts General Hospital, Clinicopathologic Conference (Published in *N Engl J Med*), Harvard Medical School.

PLATFORM PRESENTATIONS:

Michaels PJ, Brachtel EF, Bounds BC, Brugge WR, and Pitman MB. Intraductal Papillary Mucinous Neoplasm (IPMN) of the Pancreas: Cytologic Analysis and Correlation with Histologic Grade. Annual Meeting of United States and Canadian Academy of Pathology. March 2004. Vancouver, British Columbia, Canada.

Michaels PJ, Kobashigawa J, Laks H, Azarbal A, Espejo ML, Chen L, and Fishbein MC. Differential Expression of RANTES Chemokine and Leukocyte Phenotype in Acute Cellular Rejection and Quilty B Lesions. 20th International Society of Heart and Lung Transplantation Annual Meeting. April, 2000. Osaka, Japan.

Kakkis JL, **Michaels PJ**, Ma JP, Ke B, Kupiec-Weglinski J, Imagawa DK, and Busuttil RW. Pravastatin Prolongs Rat Survival after Orthotopic Liver Transplantation by Decreasing the Expression of β 2-Glycoprotein-1 and Proinflammatory Cytokines. World Congress of the Transplantation Society. July 12-17, 1998. Montreal, Quebec, Canada.

POSTER PRESENTATIONS:

Michaels PJ, Bounds BC, Brugge WR, Lewandrowski K, Pitman MB. The Clinical Utility of Cyst Fluid Analysis in Conjunction with Cytological Evaluation in the Preoperative Characterization and Subclassification of Pancreatic Mucinous Cysts. 52nd American Society of Cytopathology Annual Meeting. November, 2004. Chicago, IL.

Michaels PJ, Brachtel EF, Bounds BC, Brugge WR, and Pitman MB. Intraductal Papillary Mucinous Neoplasms (IPMN) of the Pancreas: Cytologic Analysis and Correlation with Histologic Grade. Massachusetts General Hospital Clinical Research Day. June, 2004. Boston, MA.

Michaels PJ, Kobashigawa J, Espejo ML, Alejos JC, Burch C, and Fishbein MC. Humoral Rejection in Cardiac Transplantation: Recent UCLA Experience. Sixth Banff Conference on Allograft Pathology. April, 2001. Banff, Canada.

Marchevsky A, Lockhart C, Phan A, **Michaels PJ**, and Fishbein MC. Web-based Teleconferencing Techniques as Inexpensive Tools for Transplant Patients. 2000 Annual Meeting of United States and Canadian Academy of Pathology. March, 2000. New Orleans, LA.

Kakkis JL, Schmit P, **Michaels PJ**, and Thompson J. Management of Gallstone Disease During Pregnancy in the Era of Laparoscopic Cholecystectomy. The Southwestern Surgical Congress. April, 1999. Coronado, CA.

Kakkis JL, **Michaels PJ**, Ke B, Zhao D, Kato H, Imagawa D, Kupiec-Weglinski JW, and Busuttil RW. Treatment with Pravastatin Ameliorates Rejection and Improves Survival in Liver Transplanted Rats. International Congress on Immunosuppression. December 1998. Orlando, FL.

Kakkis JL, **Michaels PJ**, Gornbein J, Terasaki P, Imagawa D, Busuttil R. Multivariate Analysis of Risk Factors in 1,008 Orthotopic Liver Transplant Recipients Reveals Significant Influence of Panel Reactive Antibody on Patient and Graft Survival. Annual Meeting of the American College of Surgeons, October 1998. Orlando, FL.

Kakkis JL, **Michaels PJ**, Ma JP, Ke B, Kupiec-Weglinski J, Imagawa DK, and Busuttil RW. Pravastatin-induced Survival in Rat Orthotopic Liver Transplantation is Accompanied by Diminished Expression of β 2-Glycoprotein-1 and Proinflammatory Cytokines. American Society of Transplant Physicians. May, 1998. Chicago, IL.

Michaels PJ, Ma J, Zhao D, Imagawa D, Busuttil R, and Kakkis JL. Pravastatin Treatment is Associated with Downregulation of TGF- β and TNF- α in Liver Transplanted Rats. 1997 Short Term Training Program Poster Session. Los Angeles, CA.

Kakkis JL, **Michaels PJ**, Ma JP, Ke B, Zhao D, Imagawa DK, and Busuttil RW. Analysis of Genetic Modifications in Liver Transplanted Rats Utilizing Messenger RNA Differential display. American Society of Transplant Surgeons. May, 1997. Chicago, IL.

EXHIBIT B

PAUL J. MICHAELS, M.D.

4214 Speedway, Austin, Texas 78751
PHONE: (512) 808-6711 EMAIL: pauljmichaels@gmail.com

FOUR-YEAR PRIOR EXPERT TESTIMONIES

DEPOSTIONS

DATE

Elizabeth Dees (Plaintiff) vs. Wyeth - <i>Deposed as expert Pathology witness for Plaintiff</i>	3/16/13
Kathryn Willis (Plaintiff) vs. Wyeth - <i>Deposed as expert Pathology witness for Plaintiff</i>	3/16/13
Nikolina Bundalo (Plaintiff) vs. Jozsef Zority, M.D. - <i>Deposed as expert Pathology witness for Plaintiff</i>	4/13/13
Mike Bendfeldt and Betty Muhr-Bendfeldt (Plaintiff) vs. HRC Medical Centers, INC. - <i>Deposed as expert Pathology witness for Plaintiff</i>	4/19/16

COURT TESTIMONIES

Toshiko Okuda (Plaintiff) vs. Wyeth - <i>Testified as expert Pathology witness for Plaintiff</i>	8/20/12
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EXHIBIT C

PAUL J. MICHAELS, M.D.

4214 Speedway, Austin, TX 78751

PHONE: (512) 808-6711 EMAIL: pauljmicahels@gmail.com

SCHEDULE OF FEES

- | | |
|---|---------------------------------------|
| 1. Cognitive work, lab work, records review, meetings | \$500.00/hour |
| 2. Depositions* | \$500.00/hour |
| 3. Court Testimony** | \$3,000/half day
\$5,500/whole day |
| 4. Overnight | As above, plus per diem (\$1000.00) |

** Depositions are charged at a two hour minimum. Any cancellations less than 48 hours will be charged two hours. Paid by side taking the deposition. Due at the time of deposition. No exceptions*

*** Cancellations of court appearances, etc., occurring less than 48 hours of the event will be billed at 50% of the scheduled rate*

Exhibit D



PAUL J. MICHAELS, M.D.
BOARD CERTIFIED IN ANATOMIC AND
CLINICAL PATHOLOGY, AND
CYTOPATHOLOGY



Literature References

1. Abed H, Rahn DD, Lowenstein L, et al. Systematic Review Group of the Society of Gynecologic Surgeons. Incidence and management of graft erosion, wound granulation, and dyspareunia following vaginal prolapse repair with graft materials: a systematic review. *Int Urogynecol J* 2011;22:789-98.
2. Altman D, Vayrynen T, Engh ME, et al.; Nordic Transvaginal Mesh Group. Anterior colporrhaphy versus transvaginal mesh for pelvic-organ prolapse. *N Engl J Med* 2011;364:1826-36.
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4. Arlt GD, Lamm T, and Klosterhalfen B. Mesh removal in inguinal hernia repair. *Eur Surg* 2003;35:42-44.
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10. Clave A, Yahi H, Hammou JC, et al. Polypropylene as a reinforcement in pelvic surgery is not inert: Comparative analysis of 100 explants. *Int Urogynecol J* 2010;21:261-70.
11. Cobb WS, Kercher KW, and Heniford BT. The argument for lightweight polypropylene mesh in hernia repair. *Surg Innov* 2005;12:63-9.
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17. Delavierre D, Rigaud J, Sibert L, et al. Definitions, classifications and terminology of chronic pelvic and perineal pain. *Prog Urol* 2010;20:853-64.
18. Diwadkar GB, Barber MD, Feiner B, et al. Complication and reoperation rates after apical vaginal prolapse surgical repair: a systematic review. *Obstet Gynecol* 2009;113:367-73.
19. Elmer C, Blomgren B, Falconer C, et al. Histological inflammatory response to transvaginal polypropylene mesh for pelvic reconstructive surgery. *J Urol* 2009;181:1189-95.
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25. Gigliobianco G, Regueros SR, Osman NI, et al. Biomaterials for pelvic floor reconstructive surgery: How can we do better? *Biomed Res Int* 2015;published online April 21 2015.
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Ethicon-Related References

1. Arnaud deposition 9/25/13 772:25 to 777:16; 779:4-11
2. ETH.MESH.00870466: 2006 Expert Meeting Norderstedt
3. ETH.MESH.01782867: "Factors Related to Mesh Shrinkage" PowerPoint presentation by Kestin Spychaj
4. Hinoul deposition 4/15/12 99:09-99:25, 4/6/12 518:14-520:20, 6/26/13 175:1-176:17, 184:18-22 328:10-24
5. Owens deposition 9/12/2012 98:11 to 99:07
6. Batke deposition 08/01/13 87:12 to 88:10, 113:3 to 114:3, 257:23 to 259:13
7. Arnaud deposition 9/25/13 769:23 to 770:4
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9. ETH.MESH.05920616: 7/20/07; Chomiak, Martin to Batke, Boris; Jamieson, Gillian; Koehler, Petra; Hellhammer, Dr. Brigitte SUBJECT: Defining light weight mesh
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12. ETH.MESH.050475773
13. ETH.MESH.04015102: 3/01/12 Batke, Boris to Mayes, Casey SUBJECT: AW: AGES Pelvic Floor Conference-Gala Dinner Invitation
14. ETH.MESH.09651393: Invention Disclosure
15. ETH.MESH.05585066: "Ultrapro" PowerPoint presentation by Boris Batke
16. ETH.MESH.05916450: "Chronic Pain Prevention/future – Bioengineer's point of view"
17. ETH.MESH.00237968: "R&D Perspective – The Journey from Prolift to Prolift +M" PowerPoint presentation by Cliff Volpe
18. ETH.MESH.05479411: Heavyweight to Lightweight Meshes PowerPoint
19. Holste deposition 07/29/13 51:3 to 53:6, 55:22 to 57:4
20. Vailhe deposition 06/20/13 182:2 to 185:5
21. ETH.MESH.01774758: December 2006 email regarding TVM Group mesh design input
22. ETH.MESH.02992139: Lightning Clinical Strategy dated 11/22/06
23. ETH.MESH.05447475: Email from Dieter Engel to John Gillespie et al. re: How inert is polypropylene?
24. ETH.MESH.02227224: PowerPoint presentation dated 05/09/08 titled MGPP Thunder Decision Meeting
25. ETH.MESH.00869908: Thunder Meeting Minutes dated 8/14/07

26. ETH.MESH.09557798: 7 Year Dog Study
27. ETH.MESH.02010834 – ETH.MESH.0201854: February 16, 2011 report written by Jurgen Trzewik and Christophe Vailhe titled “Biomechanical consideration for Pelvic floor mesh design”
28. ETH.MESH.00033325: Professional Education PowerPoint presentation titled “The Science of Augmented Extracorporeal Reconstructive Pelvic Surgery” in which the “Ideal Mesh” is described.
29. ETH-65881: Gynecare Prolift IFU
30. ETH-00255: Ethicon Gynemesh PS 2006 marketing brochure
31. Scott Ciarrocca testimony 2012-03-29 00, (Page 340:9 to 340:12)
32. Aaron Kirkemo testimony 105:14-108:16
33. ETH.MESH.03924887: Meshes in Pelvic Floor Repair
34. Hinoul trial testimony *Gross, et al. vs Gynecare, et al* 01/16/13:1151:5-1156:14; 1087:1-17;1058:22-1059:3
35. ETH.MESH.00006636: Klosterhalfen B., Interim Report Mesh Explants Pelvic Floor Repair
36. ETH.MESH.02157879-02757880: Klosterhalfen B., Intermediate Report – Prolapse Mesh Explants 6/2009

Case-Specific Materials Reviewed

Depositions of Medical Providers

Edward B. Tieng, M.D. (04/18/16)
Kimberly Ann Roberts, M.D. (04/19/16)

Depositions of Parties

Sandra Childress (03/01/16)
Timothy Childress (03/01/16)

Medical & Billing Records

Contemporary Women's Health
Blount Memorial Hospital
Ft. Sanders Regional Medical Center
Fort Sanders Obstetrical & Gynecological Group, P.C.
Innovative Pathology Services
Donald Watters, M.D.
Tennessee Urology Associates
CVS Pharmacy
Blue Cross Blue Shield of Tennessee
Parkwest Medical Center
Starr Regional Medical Center – Athens
UT Pain Clinic University of Tennessee Anesthesia Interventional Pain Clinic
TN Brain and Spine the Center for Advanced Medicine
Knoxville Comprehensive Breast Center
Fort Loudon Medical Center
University of Tennessee Medical Center
PCA Southeast
LabCorp
Neurosurgical Associates, P.C.
Knoxville Dermatology Group
Jabo's Pharmacy
Thompson Comprehensive Breast Cancer